THE INFLUENCE OF VENTRICULAR HYPERTROPHY UPON THE CARDIOGRAM OF ANTERIOR CARDIAC INFARCTION

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The presence of an S wave, preceded by an r wave in lead V5 (rS pattern) has been interpreted as indicating extreme clockwise rotation of the electrical position of the heart around its vertical axis (Goldberger, 1947, 1949). A deep S wave in V5 occurs in right ventricular hypertrophy (Myers *et al.*, 1948), while an R/S ratio less than unity in V5 (rS pattern) when occurring in conjunction with a qR pattern in VR, was considered by Pagnoni and Goodwin (1952) to be diagnostic of right ventricular hypertrophy in adults, in the absence of cardiac infarction, ventricular aneurysm, or right bundle-branch block. The rS pattern in V5 has also been shown to occur in combined ventricular hypertrophy (Goldberger, 1949; Pagnoni and Goodwin, 1952). A prominent S wave (not exceeding the R wave in amplitude) can occur in normal persons (Myers *et al.*, 1947). In Leatham's (1950) series of 100 normal subjects, the rS pattern occurred only in two, and R was equal to S in one other.

Although cardiograms have been published showing rS patterns in V5 in cases with autopsy proof of anterior infarction, (Myers *et al.*, 1949; Levy and Hyman, 1950) little has been written about this pattern in cardiac infarction.

The unexpected finding of anterior cardiac infarction at autopsy, in patients whose cardiograms showed no unusual features other than an rS complex in V5, prompted the present investigation into the genesis of this pattern, with special reference to right ventricular hypertrophy, combined ventricular hypertrophy, and cardiac infarction.

Case Material and Methods

Cardiograms were analysed from 85 adult patients with autopsy evidence of cardiac infarction, right ventricular, or combined ventricular, hypertrophy. Only cases showing a predominantly negative deflection in lead V5 were included but the study was not confined to rS patterns, cases with Qr and qrS patterns being also included.

Multiple-lead cardiograms were available, the leads examined being VR, VL, VF, and V1, V3, V5. In many cases lead V4R and leads V6 and V7 were also studied. Conventional standardization of 1 cm. = 1 mv. was employed.

Pathological Criteria

The criteria for ventricular hypertrophy previously reported by Pagnoni and Goodwin, 1952, and by Camerini *et al.* (1956), were employed, but no attempt was made to differentiate degrees of hypertrophy, since the presence of cardiac infarction in many cases rendered this impossible. Thickness of the outflow tract of the right ventricle of more than 5 mm. was considered to represent right ventricular hypertrophy, while thickness of the left ventricular muscle of more than 15 mm. was considered to represent left ventricular hypertrophy. Dilatation was also taken into account. All hearts had been examined for the presence and site of infarction.

Results

The cases were divided into six groups, according to the pathological findings, as follows.

Group	Necropsy findings	No. of cases
1	Lone right ventricular hypertrophy (RVH)	31
2	Right and left ventricular hypertrophy (R and LVH)	20
3	Infarction with lone right ventricular hypertrophy (RVH+I) ..	7
4	Infarction with lone left ventricular hypertrophy (LVH+I)	7
5	Infarction with combined ventricular hypertrophy (R and LVH+I) ..	12
6	Cardiac infarction without ventricular hypertrophy (I)	8
	Total ..	85

In Group 1, restriction to cases showing an rS pattern or a mainly negative wave in V5 eliminated many cases with autopsy and cardiographic evidence of right ventricular hypertrophy, while in Group 2, the same restriction resulted mainly in the inclusion of cases with greater hypertrophy of the right than of the left ventricle.

The site and other pathological data of the patients with infarction are shown in Table I.

TABLE I

Types of Myocardial Infarction in 34 Patients

Site of infarct	No. of cases	Other features
Anterior and septal..	25	Involvement of posterior wall in 5. Cardiac rupture in 3 (2 septum, 1 anterior left ventricle). Cardiac aneurysm in 4
Pure anterior ..	4	—
Subendocardial ..	2	Cardiac rupture in 1
Posterior—small ..	1	Cardiac aneurysm in 1
—large ..	2	—
Total	34	—

Cardiographic Analysis. In the analysis of all groups special attention was paid to the following points, having regard to the conventional criteria for right ventricular hypertrophy (Myers *et al.*, 1948; Camerini *et al.*, 1956), for combined hypertrophy (Pagnoni and Goodwin, 1952), and for cardiac infarction (Wilson *et al.*, 1944, 1947; Bain and Redfern, 1948):

Voltages of R and S waves in V4R, V1, V3, and V5.
R/S ratio in V4R, V1, V3.
S–T segment elevation in V4R, V1, V3, and V5.
T wave inversion in V4R, V1, V3, V5.
The position of the heart. (Defined as vertical if lead VL showed a dominant negative deflection, and horizontal if a dominant positive deflection.)
Dominant R wave in lead VR.
The presence of P waves suggesting right atrial hypertrophy. (Right atrial P waves: pointed P waves of 2·5 mm. or more.)
The incidence of supraventricular arrhythmia.

Prominent Q waves in leads VL, VF, and præcordial leads, indicative of infarction.
Splintering of the QRS complex in præcordial leads (qrS pattern), suggestive of infarction.
The presence of bundle-branch block.

Cardiographic Signs in Groups 1 and 2. There was no significant difference between the cases with lone right ventricular hypertrophy and those with combined hypertrophy, which is not surprising as in the latter group the right ventricle dominated the left. All cases had a dominantly negative deflection, and the majority an rS complex, in V5.

There was wide variation in the amplitude of R and S waves in the præcordial leads studied, and no conclusions could be drawn. The R/S ratio in V4R and V1, exceeded unity in less than half the cases, and there was no difference between the two groups in V4R, although in V1, the ratio was more often in excess of 1 in the cases with lone right, than in those with combined, hypertrophy (Fig 1, 2, and 3).

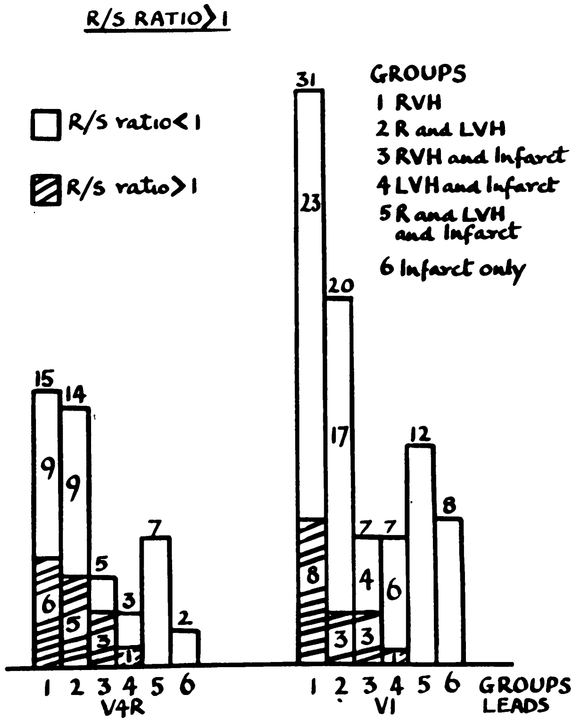

FIG. 1.—Histograms, showing the ratio of R to S waves (R/S ratio) in leads V4R and V1 in all groups.

The S–T segment was iso-electric in V1 and V5 in the majority of cases in both groups; when it was elevated, the elevation never exceeded 2 mm. in lead V5 with the exception of one case with right bundle-branch block due to lone right ventricular hypertrophy, in which the S–T segment was 4 mm. elevated (Table II, Fig. 4).

The T waves were inverted, flat, or biphasic in the majority of cases in both groups in V4R and V1, and positive in the majority in V5 (Table II).

Splintering of the QRS complex in præcordial leads (QRS pattern) was seen in only one case, a patient with lone right ventricular hypertrophy, in lead V3 (Fig. 2).

Tiny q waves preceding an rS complex in V5 were found in two cases with lone right ventricular hypertrophy.

The position of the heart was vertical in the majority of cases in both Groups 1 and 2, being slightly more frequent in Group 2 (Fig. 2 and 3).

A "right atrial" P wave was present in a minority of cases in both groups (Fig. 2).

Three cases in Group 1 and one in Group 2, had right bundle-branch block.

A dominant R wave in lead VR was seen in 13 cases (42%) in Group 1 and 8 cases (40%) in Group 2.

The Form of the Ventricular Complex in Leads V3 and V5 in the 34 Cases with Infarction. The ventricular complex in V5 was of three types. *Type A* was typical of infarction with wide deep Q waves, elevated S–T segment, and inverted T waves (15 cases). *Type B* (4 cases) was highly

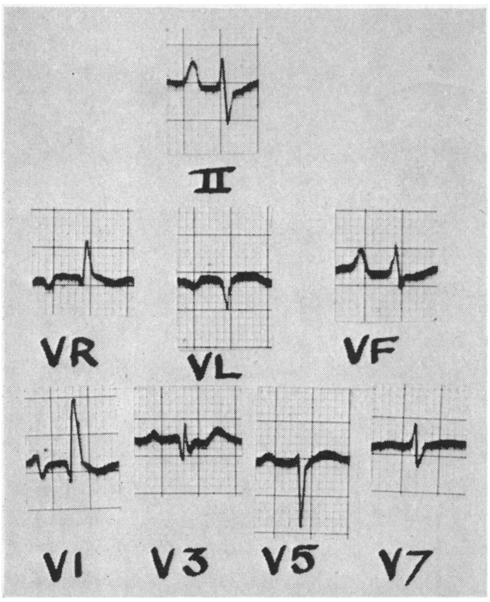

FIG. 2.—Cardiogram of patient with lone right ventricular hypertrophy without cardiac infarction. The P wave in lead II is 3 mm. in height and pointed. VR shows a qR pattern. V3 shows a QRS pattern with a deep Q wave suggestive of septal infarction. V5 shows an rS pattern. The heart is vertical in position.

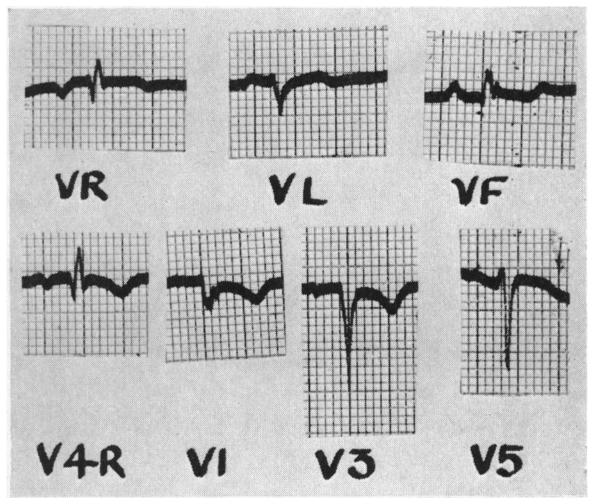

FIG. 3.—Cardiogram of patient with combined ventricular hypertrophy. There is a deep S wave in V5 with inversion of the T wave; qR in V4R, and rSR in VR. The heart is vertical in position.

TABLE II

S–T SEGMENT AND T WAVE CHANGES IN V5 IN 85 CASES

	Group	S–T elevation		T wave flat, biphasic, or inverted		Total patients
Hypertrophy only	1 2	18	35%	17	33%	51
Hypertrophy and infarction	3 4 5	17	68%	8	31%	26
Infarct only	6	7	88%	5	63%	8

suggestive but not diagnostic, and consisted of very small or "embryonic" r waves in V5 (which were smaller than those in V3), flattening or inversion of the T wave, and slight S–T elevation. Resemblance to right ventricular hypertrophy was seen in the form of a qR pattern in VR in some cases. *Type C* was frankly equivocal and consisted of an rS pattern with negative, flat or positive T

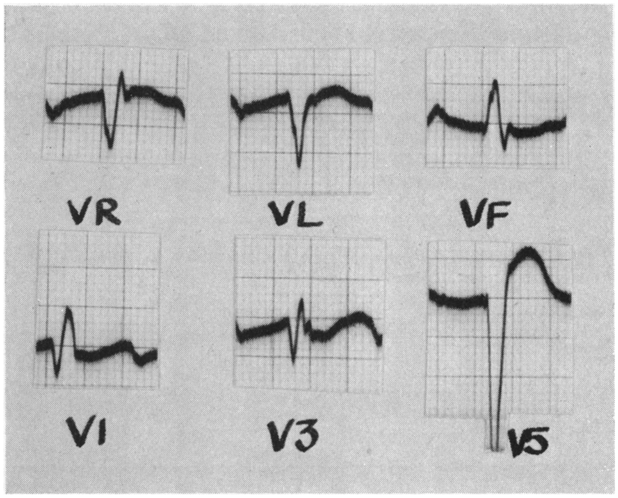

FIG. 4.—Cardiogram of patient with lone right ventricular hypertrophy without infarction. Right bundle-branch block is present, and V5 shows a qS pattern, with S–T segment elevation of 4 mm, suggesting infarction.

wave, and S–T elevation not exceeding 2 mm. This was often the only abnormality in the cardiogram, which therefore resembled that of lone or dominant right ventricular hypertrophy. Fig. 5 shows diagrammatically examples of the types of complex found, and Fig. 6A, B, and C illustrates typical cardiograms.

Type A was more frequently found in cases with infarction without ventricular hypertrophy and the converse was also true. Table III sets out the infarct groups (3, 4 5, and 6) in relation to the cardiographic types A, B, and C.

If all the cases with associated ventricular hypertrophy are grouped together and compared with those with infarct alone, the χ^2 test gives a figure of 4·05, which, when corrected, falls to 2·58, which is below the significance level, but nevertheless shows some trend which suggests that associated ventricular hypertrophy modifies the characteristic pattern of anterior infarction.

TABLE III

TYPES OF CARDIOGRAM AMONG ALL GROUPS WITH INFARCTION

Infarct groups	Cardiographic type		
	A	B+C	Total
3 RVH+I	2 }	5 }	7 }
4 LVH+I	2 } 9	5 } 17	7 } 26
5 R and LVH	5 }	7 }	12 }
6 I only	6	2	8

Site of the Infarct and Cardiographic Type. Antero-septal infarcts were present in 26 of the 34 patients; in 12 of them the cardiogram was of Type A, and in 14 of types B, or C (Table IV).

The Size of the Infarct. It was not possible to grade the size of the infarct with accuracy, but it was regarded as large when it involved an appreciable portion of the anterior wall and extended into the septum or posteriorly. Of 16 large infarcts 9 were associated with classical signs of the disease (Type A) and 7 with the rS patterns (Types B and C). Of the 18 less extensive infarcts 6

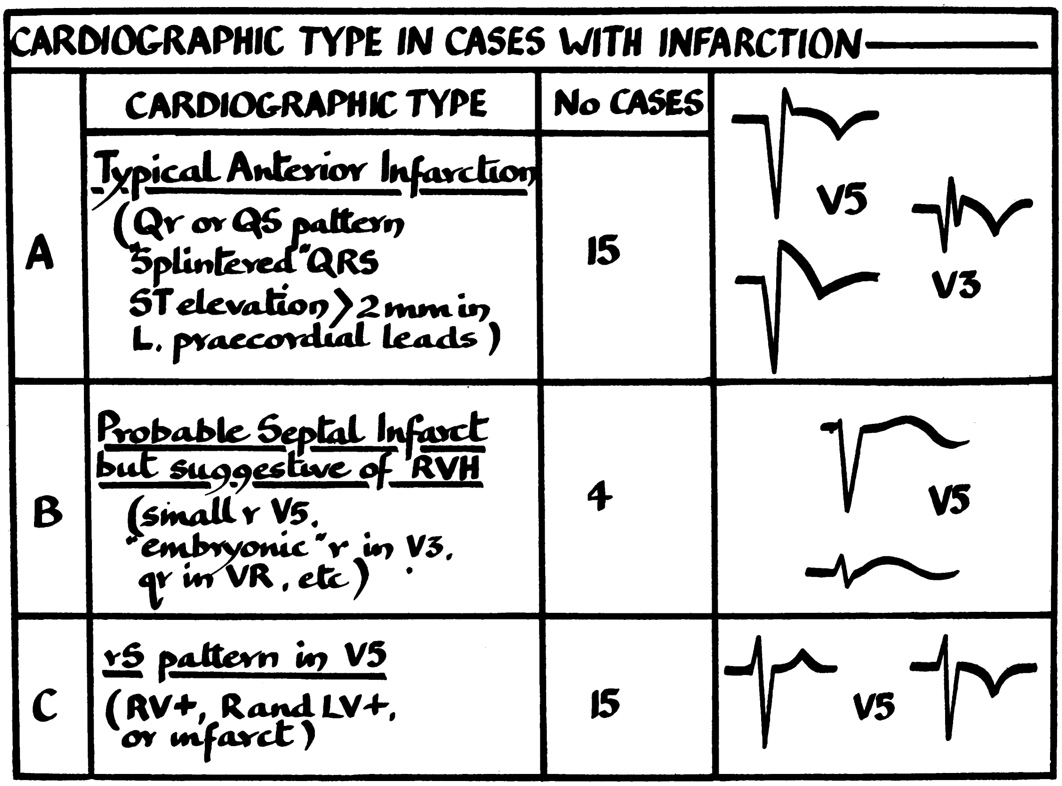

FIG. 5.—Diagrammatic table illustrating the types of complex in lead V5 in 34 cases with infarction. In cardiographic type A (typical anterior infarction) out of 15 cases, two had equivocal præcordial leads but a diagnostic pattern in VL and VF respectively.

TABLE IV
RELATION OF CARDIOGRAM TO SITE OF INFARCT IN 34 PATIENTS

Site of infarct	Cardiographic type		
	A	B+C	Total
Antero-septal	12	14	26
Antero-lateral ..	1	2	3
Posterior	1	1	2
Subendocardial ..	0	2	2
Scattered fibrosis with ventricular aneurysm	1	0	1
Total	15	19	34

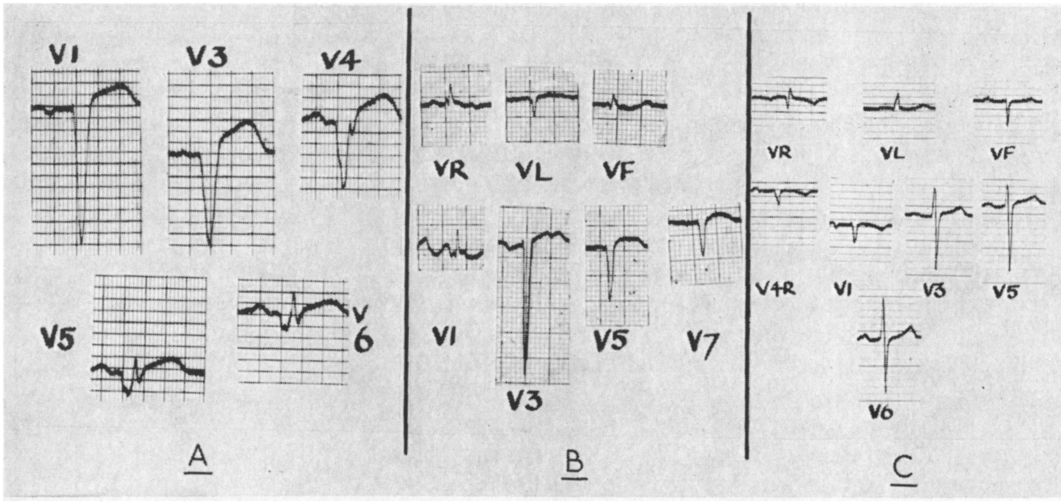

FIG. 6.—(A) *Classical infarction pattern* (*Type A. Fig. 5*) in a patient with anterior infarction without ventricular hypertrophy. There is a Qrs pattern in V5 with elevation of the S–T segment. Leads V3, 4, 6, also show evidence of infarction (each large square=0·1 sec.). (B) *Equivocal pattern* (*Type B. Fig. 5*) in a patient with lone right ventricular hypertrophy and anterior infarction. The dominant R in VR and V1, and the vertical position of the heart indicate right ventricular hypertrophy, and the rS pattern in V5 is in harmony. However, the reduction in size of the r wave from V3 to V7, and the S–T elevation suggests the possibility of infarction (each large square=0·2 sec.). (C) *rS pattern* (*Type C. Fig. 5*) in a patient with combined ventricular hypertrophy and anterior infarction. There is nothing to suggest the presence of infarction other than the association of the rS pattern in V5 with a horizontal heart, which could be due merely to the combined hypertrophy. The infarction is thus concealed (each large square=0·1 sec.).

had the classical pattern and 12 the rS pattern. Equivocal signs were therefore more common when the lesion was restricted, but large infarctions were frequently associated with the rS pattern in V5.

There were two instances of subendocardial infarction in the series and both were associated with equivocal cardiographic patterns (Table IV).

An attempt was made to determine the influence of the age of the lesion on the cardiographic pattern; but this proved difficult because of the presence of multiple infarcts, both old and recent, in some cases. There did not, however, appear to be any relation between the age of the infarct and the cardiographic type; but as will be seen later, healing was associated with the development of an rS pattern in lead V5 (Fig. 7, A, B).

Cardiographic Findings in the Cases with Infarction (*Groups 3 to 6*)

There were no significant differences between the amplitudes of R and S waves in V4R, V1, V3, and V5 in these groups and in Groups 1 and 2.

Lead V4R was recorded in 5 of the 7 cases in Group 3 (infarct and RVH) and in 3 of these the R/S ratio was greater than unity. The ratio in V1 was greater than unity in 3 of the 7 cases in the same group (Fig. 6B). In Group 4, the ratio was greater than unity in two cases, one in V4R and one in V1 (Fig. 1). It was never greater than unity in Groups 5 and 6 in either lead (Fig. 7).

The S–T segment was iso-electric in V1 in the majority of cases in all groups except Group 4 (LVH and infarct). In V5 there was elevation of the S–T segment in the majority of cases in all infarct groups. This contrasts with the presence of S–T segment elevation in the minority of cases in Groups 1 and 2 (RVH, R, and LVH). The S–T elevation was most frequent in cases with infarction only (Table II). No conclusions could be drawn about individual cases. The degree of S–T elevation was greater in the groups with infarction than in those with hypertrophy alone. Thus the maximum S–T elevation in infarction was 7 mm. in V3, and 5 mm. in V5, as compared with 4 mm. in V3, and 2 mm. in V5 in the hypertrophy cases, with the exception of the patient with bundle-branch block (Fig. 4 and 8A, B, and C).

In lead V4R or V1 the T waves were usually positive in the infarction cases, instead of negative, flat, or biphasic as in those with hypertrophy. In lead V5, however, the T wave was positive in the majority of cases of all groups, except Group 6 (Table II). This was true even when lone left ventricular hypertrophy was associated with infarction, when T wave inversion might especially have been expected in a left præcordial lead. The presence of T wave inversion in V5 usually suggested infarction (Fig. 9B), but inversion of the terminal portion of the T wave occurred in one instance with lone right ventricular hypertrophy (Fig. 9A).

Right bundle-branch block was found once in Group 3, left bundle-branch block once in Group 4 and twice in Group 5, and intraventricular block once in Group 3 and once in Group 4.

The position of the heart was vertical in only 4 of the 34 cases (12%) with infarction (three in Group 3 and one in Group 4), as compared with 34 of the 51 (66%) with hypertrophy alone.

A right atrial P wave was not found in any patient with infarction, although it occurred in 13 of the 51 who had hypertrophy alone (Fig. 2). A dominant R wave in VR was found in 3 out of 7 cases in Group 3, 3 out of 5 in Group 4 (VR was not recorded in one instance), 3 out of 12 patients

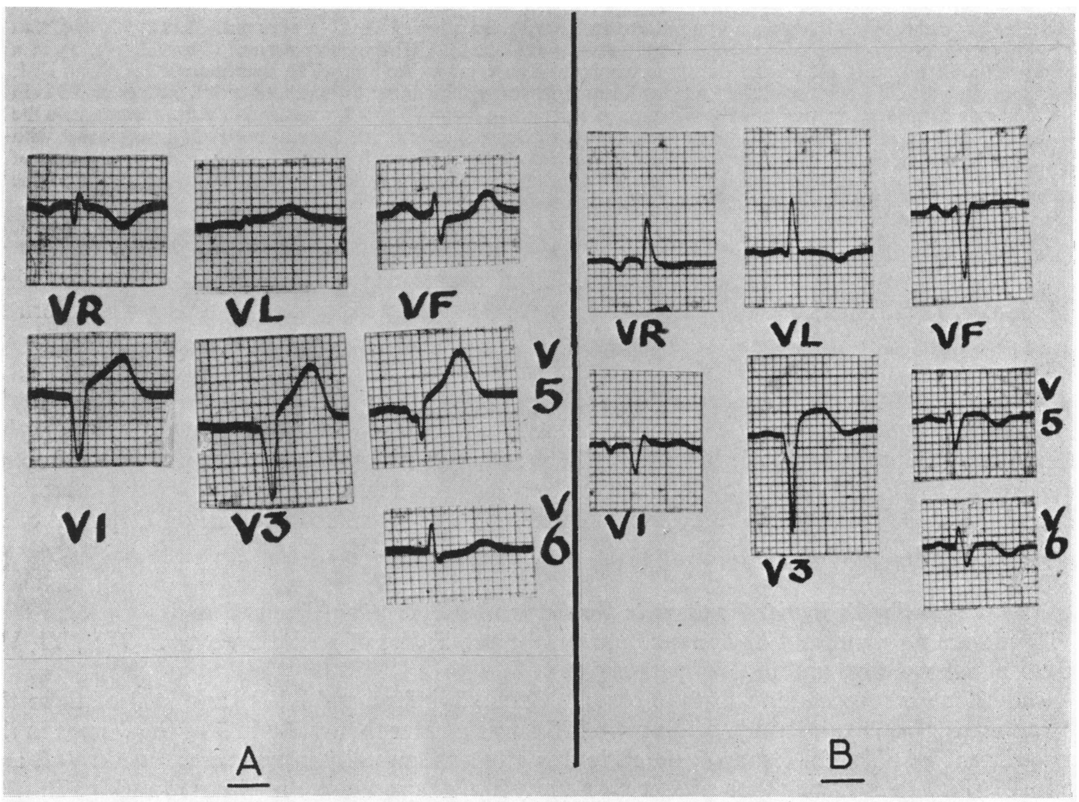

FIG. 7.—(A) Cardiograms from a patient with acute anterior infarction. There is S–T elevation in leads VL, V1, V3, and V5, with positive T waves. A rudimentary secondary r wave can be seen in V5. (B) 7 days later, the rudimentary r in V5 has grown, the q has disappeared, the S–T elevation is less, and the T wave has become inverted. There is also T wave inversion in V3–6, and VL. VR now shows a dominant R wave. The appearances are compatible with right ventricular hypertrophy. At autopsy there was occlusion of the descending branch of the left coronary artery with extensive anterior infarction, and early aneurysm formation of the left ventricle.

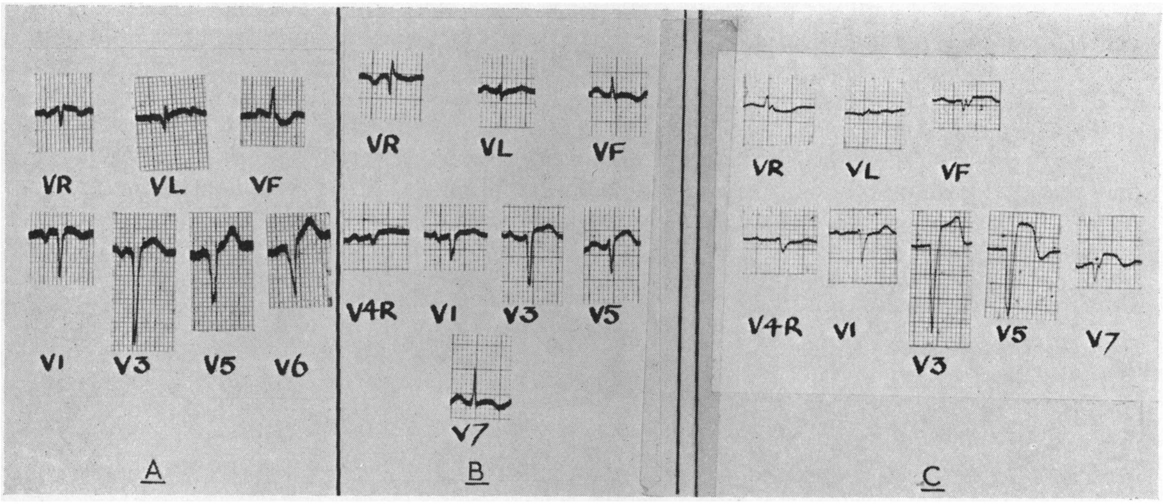

FIG. 8.—(A) Cardiogram of a patient with lone right ventricular hypertrophy without infarction. Leads V3–V6 show a qS pattern. The S–T segment is elevated 2 mm. in V5, and the T wave is upright. VL shows a q wave which is probably reciprocal to the R in VF. The pattern, however, is highly suggestive of infarction, but the heart is vertical in position. (B) Cardiogram of a patient with combined ventricular hypertrophy and anterior infarction. V5 shows the rS pattern, and there is S–T elevation of 2·5 mm. The heart is semi-vertical in position. (C) Cardiogram of a patient with combined ventricular hypertrophy and anterior infarction. Lead V5 shows the classical Qr infarct pattern, with S–T segment elevation of 5·5 mm. VR shows a dominant R wave, and VF a deep Q. Ventricular hypertrophy has not influenced the classical pattern of infarction.

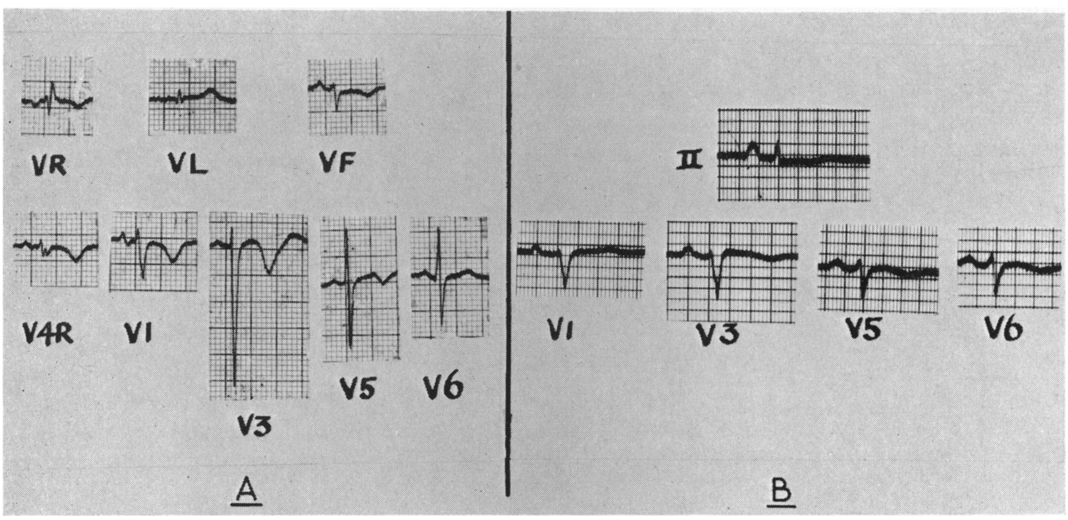

FIG. 9.—(A) Cardiogram of a patient with lone right ventricular hypertrophy, showing T wave inversion from V4R to V5. The heart is horizontal. (B) Cardiogram of a patient with combined ventricular hypertrophy and anterior infarction. The T waves are also inverted in the chest leads.

in Group 5, and 2 out of 8 in Group 6. It thus occurred even in the absence of ventricular hypertrophy and was of no value in distinguishing hypertrophy from infarction. The dominant R in VR occurred in only 3 of the 5 cases with ventricular aneurysm and was presumably reciprocal to the dominant negative deflections in left præcordial leads. These findings (Fig. 10) support those of East and Oram (1952), who showed that aneurysm can occur in the absence of this sign, in spite of the views of Goldberger and Schwartz (1948).

The important differential signs between infarction and hypertrophy were right atrial P waves and a vertical position of the heart, a combination that did not occur in infarction without hypertrophy. Right atrial P waves were also absent when infarction was accompanied by hypertrophy, and a vertical heart was seen only in 12 per cent of these cases, in contrast to 66 per cent when hypertrophy alone was present.

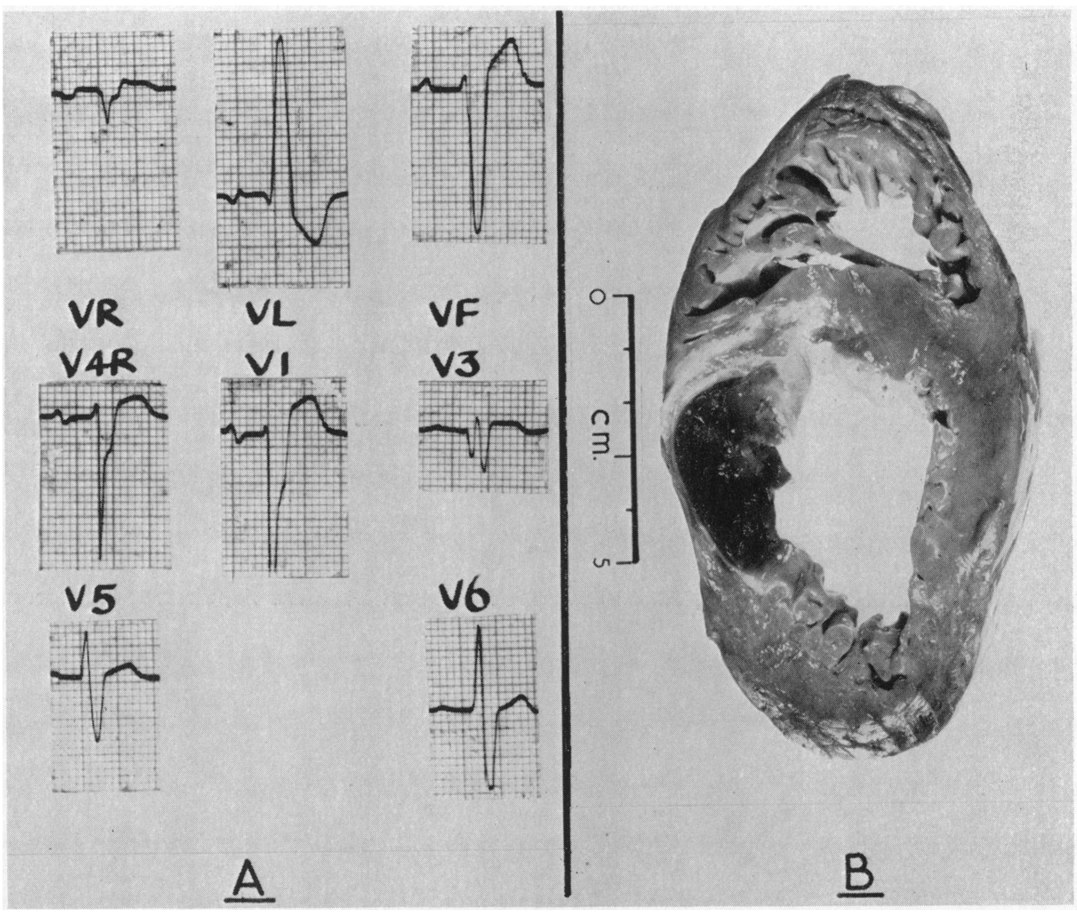

FIG. 10—(A) Cardiogram of a patient with right ventricular hypertrophy, large anterior infarction, and early cardiac aneurysm. Intra-ventricular block is present. There is a deep q in V3, but V5 and 6 show the rS pattern. The heart is horizontal. (B) Pathological specimen showing infarction, subendocardial thrombus, and early ventricular aneurysm.

Discussion and Conclusions

These results indicate that in certain circumstances the cardiographic picture of anterior cardiac infarction may be indistinguishable from that of dominant or lone right ventricular hypertrophy, and that the cardiogram of the latter condition may closely simulate that of cardiac infarction. It is clear that often the differential diagnosis cannot be made with certainty, but certain diagnostic pointers may be of value. Where there is an rS pattern in V5 there may be diagnostic evidence of infarction in other leads (V3, VL, or VF) and this should always be sought. In the absence of such evidence, a negative T wave in V5 and positive T wave in V4R, or V1, S–T segment elevation greater than 2 mm., or a small Q wave preceding the rS complex, are all suggestive of infarction. The r wave that fails to "grow" across the præcordial leads from right to left, or becomes smaller, is of course a well-known sign of infarction in the septal region.

By contrast, a vertical position of the heart, and "right atrial" P waves suggest lone or dominant right ventricular hypertrophy and make infarction unlikely. A dominant R wave in VR may occur in ventricular hypertrophy with or without infarction; it may be seen with infarction alone, but is rather more frequent in its absence. When associated with infarction it does not necessarily indicate ventricular aneurysm, nor is it essential for this diagnosis. An R/S ratio greater than unity in V4R, or V1, positive T wave in V5 and negative in V4R or V1, and S–T elevation less than 2 mm. in V5 all support hypertrophy rather than infarction, but are by no means exclusive of the latter. The differential diagnosis can best be made by considering all the possible combinations of cardiographic signs.

It is clear that cardiograms showing the rS pattern in V5 should be interpreted with great caution, and the possibility of a concealed infarct considered. Indeed, this electrocardiogram should probably be regarded as one of the variants representing cardiac infarction, bearing in mind the fact that it may be found in the absence of infarction, and very rarely even in the apparently normal heart.

The Genesis of the rS Pattern. It is generally considered that the rS pattern in V5 represents extreme clockwise rotation of the heart around the vertical axis, the usual cause being hypertrophy of the right ventricle (Goldberger, 1947; Sokolow and Lyon, 1949; Myers *et al.*, 1948; Pagnoni and Goodwin, 1952).

The results of the present investigation show that this complex can occur under varied circumstances and suggest that the concept of clockwise rotation may be an over simplification that requires further examination. The assumption that præcordial leads record a pure pattern of the electrical events occurring in the muscle that they face has been criticized (Scherlis *et al.*, 1951; Milnor *et al.*, 1953; Grishman *et al.*, 1953; Camerini *et al.*, 1956). Wilson *et al.* (1944) pointed out that although excitation of the muscle in contact with the exploring electrode produces a much larger deflection than that of an equal muscle mass at a greater distance from it, activation of all parts of the ventricular myocardium contributes to the complex derived from any electrode. In other words, the balance of electrical forces throughout the whole myocardium, and not merely the localized activity, will effect any given electrode. The deep S wave in V5 might be explained by rotation of the horizontal loop of ventricular depolarization in right ventricular hypertrophy. (Lasser *et al.*, 1951; Grishman *et al.*, 1953; Shillingford and Brigden, 1954). Such rotation must be mediated by a number of factors, including alteration of the relative position of the two ventricles due to the increased size of the right, the magnitude of the respective forces of the two ventricles, and the direction and spread of the impulse. Alteration in position of the septum and changes in its electrical activity due to infarction may also play a part.

From the practical aspect the commonest cause of a deep S in V5 is right ventricular hypertrophy, either solitary or in association with lesser degrees of left hypertrophy. As has been shown, many cases of right ventricular hypertrophy do not show a dominant R wave in V4R or V1, and in some but not all instances, this is due to the presence of associated left ventricular hypertrophy which suppresses the right præcordial R wave. In the present series, the existence of added left ventricular hypertrophy was not associated with increased amplitude of S waves in right præcordial leads, probably because of the deep S in V5.

The factors concerned in the production of the complex in V5 in infarction require elucidation. Several possible explanations may be considered.

The Age of the Infarct and the Healing Process. It might be supposed that the rS complex represents partial healing of the infarct, with disappearance of the Q wave, but only partial "recovery" of the r wave. Fig. 7 shows that this suggestion is at least feasible, since in the second cardiogram the S–T elevation and q wave had disappeared, to be replaced by the rS pattern. However, this hypothesis does not explain the deep S wave, nor is the rS pattern confined to "old" infarcts, and other factors must be involved.

The Influence of Ventricular Hypertrophy. The results strongly suggest that hypertrophy of the right ventricle modifies the classical infarction pattern in V5. When right ventricular hypertrophy is present, the loop of the QRS vector is orientated to the right and posteriorly in the horizontal projection, while in left ventricular hypertrophy the loop is orientated to the left and posteriorly. In typical anteroseptal infarction, the loop is directed to the left, with disappearance of the right anterior initial portion of the loop due to antero septal activation. This results in Q waves from anteroseptal præcordial leads (Grishman *et al.*, 1953). It seems possible that previous rotation of the loop to the right by right ventricular hypertrophy might result in the disappearance of anterolateral negative vector forces of infarction, and thus the replacement of the q wave by a small r wave in V5. A similar explanation might hold when lone left ventricular hypertrophy is present in association with infarction, but the mechanism is obscure and further deductions cannot fairly be made.

The Site and Size of the Infarct. In the majority of patients the infarcts were anteroseptal, but subendocardial, lateral and even posterior lesions occurred in a few cases. It is probable, however, that the rS pattern in infarction in V5 depends mainly upon septal involvement, with consequent disorientation of normal septal vectors. The size of the infarct apparently has no great bearing, since both large and small ones were found in association with the rS pattern.

The thickness of the ventricular wall involved may be of some importance. rS patterns in V5 were found in the two cases with subendocardial infarction. It is known that q waves may not develop in subendocardial infarction (Prinzmetal *et al.*, 1953) while the rS pattern has been found in subepicardial infarction involving the posterolateral wall of the left ventricle by Levy and Hyman (1950), who noted that . . . "since the spread of excitation from the endocardium outward is not affected in this type of lesion, a Q wave will not appear, and only a reduction in amplitude of the R wave or the appearance of an S wave in the leads which face the infarcted area may occur. . . ."

Intramural infarction extending neither to endocardium nor epicardium may also fail to produce a characteristic Q wave. Many of the cases in the present series were studied retrospectively and it was not always possible to discover the thickness of the infarcted ventricular wall from the necropsy reports.

The findings in the series studied here suggest that the most important factors concerned with the production of an rS pattern in V5 in anterior infarction are associated ventricular hypertrophy (usually dominantly right) and changes of healing in the infarct, which may be cardiographically surprisingly rapid (Fig. 7).

The rarity of a vertical heart position in association with infarction suggests that this is an important differential diagnostic point between ventricular hypertrophy and infarction. A horizontal position does not indicate infarction, but a vertical position makes this diagnosis improbable, though it does not exclude it. Possibly the presence of anterior infarction rotates the QRS vector to the left in the frontal plane despite the presence of right ventricular hypertrophy, thus preventing the "vertical" position from developing. A right atrial P wave may possibly exclude infarction, for it was not seen in any case in this series.

The term clockwise rotation should probably be abandoned in favour of the term "rS complex in V5", which may be due to lone right ventricular hypertrophy, dominant right ventricular hypertrophy, anterior infarction, or a combination of all. Occasionally, it may even be a normal finding.

The results show not only the cardiographic resemblance of infarction to right ventricular hypertrophy, but also that of right ventricular hypertrophy to infarction (Myers, 1950). Fig. 2 shows a prominent Q in V3, with rS in V5, apparently diagnostic of septal infarction, yet none was found at necropsy. Clearly, alteration of the position of the two ventricles, of the septum, and of the balance of forces due to right ventricular hypertrophy, may re-orientate the projection of the QRS vector in the horizontal plane in a fashion similar to that produced by septal infarction, thus counterfeiting the cardiogram of the latter.

Summary

The causes of an rS complex in lead V5 have been investigated by the study of necropsies on 85 patients all of whom had a predominantly negative deflection in that lead.

A predominantly negative deflection in V5 was seen in lone right ventricular hypertrophy, in combined ventricular hypertrophy, and in hypertrophy with anterior cardiac infarction. The typical patterns of anterior infarction were less common when ventricular hypertrophy was present than when infarction occurred in the absence of right ventricular hypertrophy. Close resemblance was found between the electrocardiogram of right ventricular hypertrophy and that of anterior infarction. The differential diagnosis is discussed, and also the reason for the replacement of the classical infarct pattern in V5 by the rS pattern.

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